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Office of Administrative Law Judges
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CASE NO.: 1999-BLA-01260

In the Matter of

ODESSA JONES,
Widow of CLELL M. JONES,
Claimant,

v.

ENERGY WEST MINING COMPANY,
Employer,

and

ENERGY MUTUAL MINING COMPANY,
Carrier,

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS
Party-in-Interest

Appearances:

Jonathan Wilderman, Esquire
For Claimant

Ronald Gilbertson, Esquire
For Employer/Carrier

Before: **PAUL H. TEITLER**
Administrative Law Judge

DECISION AND ORDER GRANTING BENEFITS

This proceeding arises from a claim for benefits under the Federal Coal Mine Health and Safety Act, 30 U.S.C. §§901-945 (the Act) and the regulations issued thereunder, which are found in Title 20 of the Code of Federal Regulations. Benefits under the Act are awardable to coal miners who are totally disabled within the meaning of the Act due to pneumoconiosis. Benefits are also awardable to the survivors of persons whose death was due to pneumoconiosis. Pneumoconiosis is a dust disease of the lungs resulting from coal dust inhalation and is commonly known as black lung.

This case was referred to the Office of Administrative Law Judges by the Director, Office of Workers' Compensation Programs, for a formal hearing. The hearing was held before me in Price, Utah on October 25, 2000. At that time, all parties were afforded full opportunity to present evidence and argument. The record was left open after the hearing to permit the parties to submit post-hearing briefs.

The findings of fact and conclusions of law which follow are based upon my observations of the appearance and demeanor of the witnesses who testified at the hearing and upon my thorough analysis and review of the entire record, arguments of the parties, and applicable statutes, regulations, and case law. Each exhibit entered in evidence, although possibly not mentioned in this Decision, has been carefully reviewed and considered in light of its relevance to the resolution of a contested issue. The resolution of black lung benefit claims frequently requires the evaluation and comparison of conflicting evidence. Where evidence may appear to conflict with the conclusions in this case, the appraisal of the relative merits and evidentiary weight of all such evidence was conducted strictly in accordance with the quality standards and review procedures set forth in the Act, regulations, and applicable case law.

ISSUES

The parties have stipulated that Mr. Jones was employed by Energy West or one of its components and had 18 ½ years of qualifying coal mine employment. Therefore, the remaining issues presented for resolution are:

(1) Whether Mr. Jones had pneumoconiosis.

(2) Whether Mr. Jones' pneumoconiosis arose from his coal mine employment.

(3) Whether Mr. Jones' death was hastened by pneumoconiosis.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Procedural History

Clell Jones (Mr. Jones) filed an application for Federal Black Lung Benefits on November 19, 1986, but withdrew this claim on May 20, 1988. Mr. Jones died on June 6, 1998. His widow, Odessa Jones[1], (the Claimant), filed a claim for survivor's benefits on January 8, 1999. (DX 1)[2]. Employer controverted this claim on March 22, 1999. (DX 14). Claimant then requested a formal hearing, and a hearing was held before the undersigned on October 25, 2000.

Title 20 C.F.R. Part 718 is the controlling law in this case because Claimant filed for benefits under the Act after January 1, 1982. On January 19, 2001, however, the U.S. Department of Labor published new regulations pertaining to the Act. Many of these new regulations, particularly those under Part 718, are applicable to claims pending before the Office of Administrative Law Judges. On February 9, 2001, the United States District Court for the District of Columbia issued a Preliminary Injunction Order, No. 1:00CV03086, *Mining Associates, et al, v. Chao, et al,* which stays the implementation of many of the new regulatory provisions.

In compliance with the Preliminary Injunction Order, on February 26, 2001, the undersigned issued an Order directing the parties to submit briefs "stating with specificity how application of the amended regulatory provisions at 20 C.F.R. §§ 718.104(d), 718.201(a)(2), 718.201(c), 718.204(a), 718.205(c), and 718.205(d) will affect the outcome of this claim." Claimant, Employer, and Director submitted briefs. Upon review of the facts of this case, I find that the amended regulations will not affect the outcome of this case because Claimant is entitled to benefits under both the amended regulations and the previous regulations as they existed before the amended regulations were introduced.

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[1] Mrs. Jones was born on April 30, 1921. She married Mr. Jones on August 20, 1948 and has not remarried since his death. Mrs. Jones is his only dependent for the purpose of possible benefit augmentation under the Act.

[2] The following references will be used herein: "CX" designates Claimant's exhibits; "DX" designates Director's exhibits; "RX" designates Employer's exhibits; and "TX" designates pages from the transcript of the hearing held before me on October 25, 2000.

The application of amended §718.104(d), which provides that a treating physician's medical opinion is entitled to greater weight, will not affect the outcome of the instant case because this section applies only to evidence "developed in connection with benefit claims after the effective date of the final rule." 65 Fed. Reg. 79933. The effective date of the amended regulations is January 19, 2001. Therefore, because Mr. Jones' treating physician, Dr. Pearl, did not gather any medical evidence after that date, this amended regulation will not affect the outcome of this case.

The application of amended §718.201(a) will not affect the outcome of the case. Section 718.201(a) adds the terms "clinical" and "legal" pneumoconiosis and clarifies that both restrictive and obstructive lung disease may arise from coal mine employment. Although the terms "legal" and "clinical" pneumoconiosis do not explicitly appear in the former regulations, in essence the language of the former regulations provided for them. Section 718.201 of the former regulations provides:

> For purposes of the Act, *pneumoconiosis* means a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. This definition includes but is not limited to, coal workers' pneumoconiosis, anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, progressive massive fibrosis, silicosis or silicotuberculosis, arising out of coal mine employment. For purposes of this definition, a disease "arising out of coal mine employment" includes any chronic pulmonary disease resulting in respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment.

20 C.F.R. §718.201.

As the plain language of the regulation states, the list of impairments given in the definition is not an exhaustive list. Accordingly, pulmonary or respiratory impairments that do not appear in this list, but are found to have arisen out of the miner's coal mine employment, may fall within the definition of pneumoconiosis. Therefore, because the former regulation is broad and allows for disorders other than those listed to be included within the definition of pneumoconiosis, including chronic restrictive or obstructive pulmonary disease arising out of coal mine employment, I find that the application of the amended §718.201(a)(2) will not affect the outcome of the case. Moreover, the addition to the regulation at §718.201(c) stating that pneumoconiosis is a "latent and progressive disease" is a codification of case law as it currently exists in the Tenth Circuit. *See Lukman v. Director, OWCP,* 896 F.2d 1248, 1253 (10th Cir. 1990); *Director, OWCP v. Gurule,* 653 F.2d 1368, 1371 (10th Cir. 1981). Accordingly, I find that the amended regulations found in §718.201 will not affect the outcome of the case.

The application of amended §718.205, which adopts the hastening death standard, will not affect the outcome of the case. This addition is merely a codification of Tenth Circuit case law. *See Northern Coal Co. v. Director, OWCP,* 100 F.3d 871, 874 (10th Cir. 1996) (Survivors are entitled to benefits if pneumoconiosis hastened the miner's death "to any degree"). Accordingly, because the hastening death standard is already the standard in the Tenth Circuit, application of this amended regulation will not affect the outcome of the case.

In addition, Employer has objected to the introduction of Claimant's medical report from David S. James, M.D. dated February 11, 2001 as untimely. Claimant has likewise objected to a medical report from Dr. Richard L. Naeye, M.D. dated October 5, 2000 and a medical report from Peter F. Tuteur, M.D. dated November 9, 2000 as untimely. However, §725.456(b)(2) allows the judge, at his discretion, to admit documentary evidence which is late if the parties agree or if good cause is shown. *Newland v. Consolidation Coal Co.,* 6 B.L.R. 1-1286 (1984). The parties discussed these medical reports at the hearing on October 25, 2000. Moreover, given the complexity of the medical issues involved, I find that there is good cause to admit the medical reports of Drs. James, Naeye, and Tuteur.

Testimony of Ms. Odessa Jones.

Claimant testified at the hearing held on October 25, 2000 as follows (TX at 30-50): When Mr. Jones returned home from work in the coal mine, his clothes were dusty and black and you could see coal dust in his nostrils, ears, and eyebrows. (TX at 35). Her husband told her that he worked in an environment full of coal dust. (TX at 36). In addition, Mr. Jones quit smoking in 1948 when they were married and that there was no smoking in their home. (TX at 46).

Mr. Jones' main position in the coal mine was that of shuttle car operator. (TX at 36). This work required him to go close to the face to pick the coal that was loaded from the continuous miner machine. (TX at 36). In addition, from 1949 to 1953, Mr. Jones worked in a very dusty environment where they were blasting and shooting coal in order to mine it. (TR at 38). Mr. Jones retired on or about August 31, 1985 at the age of 63 due to breathing problems. (TX at 38-39). He did not work at all after he retired. (TX at 39).

Within a year of retiring, Mr. Jones filed for Federal Black Lung benefits. (TX at 39). In the mornings, he would cough and spit up. (TX at 39). He spent most of his time watching television and was not able to do physical activities because of his difficulty breathing. (TX at 43). He was on supplemental oxygen for the last two years of his life. (TX at 40). Mr. Jones' condition continued to worsen until the time of his final hospitalization. (TX at 40). In early May 1998, Mr. Jones was taken to Castleview Hospital because he became so weak he could not walk to the bathroom. (TX at 41). He was transferred to Later Day Saints(LDS) Hospital where he remained until his death on June 6, 1998. (TX at 43). During that time he was on mask oxygen on a full-time basis. (TX at 43-44).

Testimony of Collier Pierce

Collier Pierce testified on behalf of the Claimant at the hearing on October 25, 2000 as follows (TX at 50-64): Mr. Pierce was Mr. Jones' co-worker from May 1972 until August 1985. (TX at 51-52). He observed Mr. Jones working close to the face as a shuttle car operator where he was exposed to heavy concentrations of dust from mining and loading coal. (TX at 52-53). The dust was so heavy you could not see the other miners because of it. (TX at 54). Moreover, the mine Mr. Jones and Mr. Collier worked in was a dry mine, which is more dusty than a wet one. (TX at 56).

Mr. Jones also did some work as a utility man in the tipple area which was dry and dusty. (TX at 56-57). He also worked as a lampman. (TX at 57). As a lampman, Mr. Jones cleaned lamps and the bathhouse. (TX at 57). Working as a lampman also exposed Mr. Jones to dust because he cleaned up after miners changing out of dusty work clothes and swept dust off the floor. (TX at 58). Mr. Jones also did roof bolting. (TX at 58-59). All the jobs Mr. Jones performed involved substantial dust exposure and required heavy lifting, walking grades, and moving equipment. (TX at 59-60).

On cross-examination Mr. Pierce stated he wore a air filter, which was a dry filter mask with elastic straps. (TX at 62). In addition, all of the face area jobs Mr. Jones performed were dustier than some of the outlying jobs. (TX at 62). The tipple job was dustier than in the mine. Mr. Pierce stated, "[t]hey had to almost drag you out there screaming and yelling to work there because of the dust." (TX at 63). A lampman job was less dusty. (TX at 63). However, Mr. Jones worked as a shuttle car operator 90 to 95 percent of the time. (TX at 64).

Testimony of Dennis Clell Jones

Dennis Clell Jones testified on behalf of the Claimant at the hearing on October 25, 2000 as follows (TX at 64-68): Mr. Dennis Jones is the son of Claimant and Mr. Jones. (TX at 65). He is a school principal, but he previously worked as a coal miner for six years. (TX at 65). At times he worked with his father at the face of the coal mine, but not at the time when the mine was drilling and shooting coal. (TX at 66). He testified that the face of the mine was dusty. (TX at 67)

Mr. Jones observed that his father's activity level continually got worse after he retired from the mine. (TX at 67). Mr. Jones had been a hunter, but he was unable to get out of the truck, could not go to the mountains, and was limited as to what he could do around the house. (TX at 67-68). In addition, he never saw his father smoke. (TX at 68).

Medical History

Mr. Jones was evaluated by Dr. Lincoln for purposes of black lung benefits on November 21, 1986. (DX 32). Dr. Lincoln reported that Mr. Jones had a chronic non-

productive cough for four to five years with sputum and dyspnea, but that his chest sounds were clear. Dr. Lincoln did not diagnose Mr. Jones with pneumoconiosis.

Mr. Jones was examined at Employer's request by Dr. Farney on June 12, 1987. (DX 32). Dr. Farney reported that Mr. Jones had shortness of breath which generally occurred when he was walking fast up an incline or up stairs. In addition, his evaluation states "[t]he patient claims that the shortness of breath was present when working as a bath house attendant and stated that he had difficulty performing his job accordingly." Dr. Farney's examination revealed wheezing with forced exhalation on the chest examination. However, he did not find that Mr. Jones had pneumoconiosis.

Mr. Jones went to the Castleview Hospital emergency room on March 21, 1995 because of a nose bleed. A chest exam conducted during that visit noted rhonchi in the left lung that did not resolve with coughing. He began receiving supplemental oxygen in 1996. Mr. Jones was first evaluated by Dr. Pearl on February 9, 1998. During this examination, Dr. Pearl's diagnosis was that Mr. Jones suffered from new onset interstitial lung disease of unclear etiology, history of coal dust exposure of significance, silica dust exposure and that he had "dyspnea on exertion when walking without oxygen short distances." (CX 5). Dr. Pearl also noted that bilateral inspiratory crackles were present in both lower lung fields. No clubbing of the fingers was noted in this report.

Mr. Jones was admitted to Castleview Hospital in Price, Utah on May 7, 1998 with a complaint of four to five days of increasing cough and increasing shortness of breath. The admitting diagnosis was pulmonary fibrosis and he was treated by Dr. David Nichols. His respiratory status weakened and he was transferred to LDS Hospital. The admitting diagnosis at LDS was pulmonary fibrosis, lower lobe pneumonia, and congestive heart failure. In addition, right lower extremity deep venous thrombus was found and treated with anticoagulants. An x-ray revealed pulmonary fibrosis, and possible interstitial pneumonia or pulmonary edema. Mr. Jones respiratory condition worsened and two courses of methotrexate were administered for the pulmonary fibrosis. While at LDS Mr. Jones underwent a CT-scan of the chest. Dr. Pisani read the CT-scan as negative for asbestos and pneumoconiosis because there were no calcified pleural plaques to suggest asbestos or silicosis. Mr. Jones died on June 6, 1998. The final diagnoses according to Dr. Pearl were idiopathic pulmonary fibrosis and deep venous thrombus. He did not diagnose pneumoconiosis. Mr. Jones had no significant hospitalizations prior to his final one in 1998. (CX 3). His death certificate was filed on June 17, 1998 and was completed by Dr. Pearl (DX 5). The death certificate lists respiratory failure and idiopathic pulmonary fibrosis as the causes of death.

Entitlement to Benefits: In General

Under Part 718, benefits are provided to eligible survivors of a miner whose death was due to pneumoconiosis. §718.205(a). The burden of persuasion is on the claimant to establish

each element of entitlement by a preponderance of the evidence. *Director, OWCP v. Greenwich Collieries*, 312 U.S. 267 (1994).

Issue 1. Whether Mr. Jones had pneumoconiosis.

The Benefits Review Board (the Board) has held that in a Part 718 survivor's claim, the Administrative Law Judge must make a threshold determination as to the existence of pneumoconiosis under §718.202(a) prior to considering whether the miner's death was due to the disease. *See Trumbo v. Reading Anthracite Co.*, 17 BLR 1-85 (1993). In the instant case, the question of pneumoconiosis is vigorously contested by the Employer. Accordingly all evidence will be examined.

Pneumoconiosis is defined as "a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment." §718.201; *See Northern Coal Co. v. Director, OWCP,* 100 F.3d 871 (10th Cir. 1996). This definition includes clinical and legal pneumoconiosis and includes, but is not limited to, coal workers' pneumoconiosis (CWP), anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, progressive massive fibrosis, silicosis or silicotuberculosis arising out of coal mine employment. §718.201. In addition, for purposes of this definition, a disease "arising out of coal mine employment" includes "any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment." §718.201. Moreover, "[i]t is well recognized that pneumoconiosis is often a latent, progressive and insidious disease and therefore evidence establishing total disability due to pneumoconiosis may relate backward in time to establish an earlier onset date in the absence of earlier contradictory like evidence." *Director, OWCP v. Joaquin Gurule,* 653 F.2d 1368, 1371 (10th Cir. 1981) (citing *Begley v. Mathews*, 554 F.2d 1345 (6th Cir. 1976), cert. denied, 430 U.S. 985, 987 (1977)). *See also* 20 C.F.R. §718.201.

Section 718.202 sets forth four ways to establish the existence of pneumoconiosis:

(1) X-ray evidence, or

(2) Biopsy or autopsy evidence, or

(3) Regulatory presumptions set forth in §§718.304, 718.305, or 718.306, or

(4) Physicians' opinions based upon objective medical evidence.

a. Chest X-ray Evidence

Under §718.202(a)(1), a finding of pneumoconiosis may be established by chest x-rays conducted and classified in accordance with §718.102. To establish the existence of pneumoconiosis, a chest x-ray must be classified as category 1, 2, 3, A, B, or C, according to the ILO-U/C classification system. A chest x-ray classified as category 0, including subcategories 0/1, 0/0, or 0/-, does not constitute evidence of pneumoconiosis.

The Board has held that under Part 718, where the x-ray evidence is in conflict, consideration shall be given to the readers' radiological qualifications. *Dixon v. North Camp Coal Co.*, 8 BLR 1-344 (1985). The administrative law judge may assign more weight to the x-ray interpretation of a B-reader. *Aimone v. Morrison Knudson Co.*, 8 BLR 1-32 (1985); *Vance v. Eastern Associated Coal Corp.*, 8 BLR 1-69 (1985). The Board has also held the interpretation of an x-ray by a physician who is a board-certified radiologist as well as a B-reader may be given more weight than the interpretation of a physician who is only a B-reader. *Scheckler v. Clinchfield Coal Co.*, 7 BLR 1-128 (1984). Where there is conflict among x-ray interpretations, for example, when the interpretations of two B-readers conflict, the administrative law judge, as the trier of fact, must resolve it. *Dees v. Peabody Coal Co.*, 5 BLR 1-117 (1982); *Elkins v. Beth Elkorn Corp.*, 2 BLR 1-683 (1982).

In the instant case, I have graphed 27 readings of fifteen x-rays and four readings of one CT-Scan. The following is a list of admissible x-ray readings and the names and qualifications of the interpreting physicians if they are known[3]:

Ex. No.	Date of X-ray	Date Read	Physician/ Qualifications	Interpretation
DX 21	05/10/82	05/15/82	Denman	Parenchymal calcification secondary to old inflammatory disease. 0/0.
DX 32	11/12/86	12/08/86	Sargent B, BCR	0/0
DX 21	12/03/86	12/03/86	Shipman	Mild hyperinflation of the lungs; COPD.
DX 32	05/27/87	05/27/87	Davis B	0/0

[3] The symbol "B" denotes a physician who was an approved "B-reader" at the time of the x-ray reading. A B-reader is a radiologist who has demonstrated his expertise in assessing and classifying x-ray evidence of pneumoconiosis. These physicians have been approved as proficient readers by the National Institute of Occupational Safety & Health, U.S. Public Health Service pursuant to 42 C.F.R. §37.51 (1982).

The symbol "BCR" denotes a physician who has been certified in radiology or diagnostic roentgenology by the American Board of Radiology, Inc., or the American Osteopathic Association. 20 C.F.R. §727.206(b)(2)(iii).

Ex. No.	Date of X-ray	Date Read	Physician/ Qualifications	Interpretation
DX 21	03/12/95	03/13/95	Sheya	Marked bronchial wall thickening; non-specific irregular shadows.
DX 21	07/22/96		Sheya	Increase of irregular shadows.
DX 20	02/09/98		Pisani	Extensive interstitial lung disease; large areas of honeycombing.
DX 10	05/01/98	01/25/99	Mann B	q, t, all zones, 3/3.
DX 21	05/09/98		Giuliano	Changes of pulmonary fibrosis; probably superimposition of either interstitial pneumonia or edema.
DX 21	05/10/98		Giuliano	Interstitial lung disease.
DX 09`	05/11/98		Morrison	Unspecific findings. Possible scleroderma or rheumatoid disease.
DX 10	05/11/98	01/25/99	Mann B	Q,T all zones 3/3; consistent with pneumoconiosis.
DX 28	05/11/98	06/23/99	Scott B, BCR	Negative for pneumoconiosis.
DX 28	05/11/98	06/23/99	Wheeler B, BCR	Negative for pneumoconiosis.
DX 28	05/14/98	06/23/99	Wheeler B, BCR	Negative for pneumoconiosis.
DX 28	05/14/98	06/23/99	Scott B, BCR	Negative for pneumoconiosis.
DX 10	05/14/98	01/25/99	Mann B	Q,T, all zones 3/3
DX 28	05/27/98	06/23/99	Wheeler B, BCR	Interstitial fibrosis 0/0

Ex. No.	Date of X-ray	Date Read	Physician/ Qualifications	Interpretation
CX 03	05/27/98	10/03/00	James B	T, T, all zones 2/3
DX 09	05/27/98		Booth	Interstitial lung disease
DX 10	05/27/98	01/25/99	Mann B	Q. T all zones 3/3
DX 28	05/27/98	06/23/99	Scott B, BCR	Negative for pneumoconiosis. Linear fibrosis UIP.
DX 28	05/28/98	06/23/99	Wheeler B, BCR	Interstitial fibrosis negative for pneumoconiosis.
DX 28	05/28/98	06/23/99	Scott B, BCR	Linear fibrosis negative for pneumoconiosis.
DX 10	05/28/98	01/25/99	Mann B	Q.T, all zones 3/3
DX 28	06/06/98	06/23/98	Wheeler B, BCR	Interstitial pneumoconiosis negative for pneumoconiosis.
DX 28	06/06/98	06/23/99	Scott B, BCR	Linear fibrosis negative for pneumoconiosis.

Based upon all the x-ray evidence listed in the table above and considering the qualifications of the interpreting physicians and the recency of the films, I find Claimant has not established the presence of pneumoconiosis by means of the x-ray evidence of record pursuant to §718.202(a)(1). Because the x-ray readings are in conflict, it is necessary to weigh the credentials of the doctors who interpreted them.

Dr. Mann, a "B" reader, interpreted as positive the May 1, 11, 14, and 27, 1998 x-rays. In addition, Dr. James, who is also a "B" reader, interpreted the May 27, 1998 x-ray as positive for pneumoconiosis. All of the other readings were negative for pneumoconiosis. Drs. Wheeler, Scott, and Sargent, all of whom are B Readers as well as Board Certified Radiologists, reported eleven readings that were negative for pneumoconiosis. Accordingly, the fact that there were more negative readings of the x-rays, coupled with the superior credentials of Drs. Wheeler, Scott, and Sargent, necessarily leads to the conclusion that Claimant has not established Mr. Jones had pneumoconiosis through the x-ray evidence.

CT Scan Evidence

There is one CT Scan of record dated February 9, 1998. The interpretations of this CT-Scan have been summarized in the table below:

EX. No.	Date of CT Scan	Date Read	Physician Qualifications	Interpretation
DX 20	02/09/98		Pisani	Extensive interstitial lung disease, including large areas of honeycombing.
CX 03 CT SCAN	02/09/98	10/03/00	James	Bilateral linear densities more than nodular, honeycombing positive for pneumoconiosis.
DX 28 CT SCAN	02/09/98		Wheeler B, BCR	Minimal emphysema with small bullous blebs in basal segments and upper lobes negative for pneumoconiosis.
DX 28 CT SCAN	02/09/98		Scott B, BCR	Scattered bullae and blebs, non-specific linear fibrosis, negative for pneumoconiosis.

Upon considering the qualifications of the interpreting physicians and the recency of the film, I find Claimant has not established the presence of pneumoconiosis by means of the CT-Scan evidence of record. The CT-Scan was interpreted as positive for pneumoconiosis by Dr. James and negative for pneumoconiosis by Drs. Wheeler, Scott, and Pisani.

Dr. James stated that the CT-Scan did not reveal silicosis. However, he opined that this CT scan was not necessarily inconsistent with his x-ray interpretation. (TX at 122-123).

> The main radiographic findings are honeycombing and fibrosis. There's a lack of what they say silicosis without apical distribution and what they're referring to there is the nodular disease that we see with silicosis and I would agree with that, but there isn't radiographic evidence of the nodular form of silicosis. But my and other B readings from a later date would be consistent with honeycombing and fibrosis. The rheumatoid disease is art of the differential. On the radiograph, there are no specific changes on a radiograph or CT that can say that type of condition is due to rheumatoid disease.

(TX at 124).

In addition, Dr. James said he would not expect a radiologist to find CT-Scan evidence of pneumoconiosis on his report for two reasons. First, there are a number of different disorders that can cause interstitial fibrosis and there was no evidence to suggest that the radiologist knew that Mr. Jones was a coal miner. In addition, "the presence of diffused interstitial fibrosis is not as widely discussed in the radiographic literature and so he may just not have been aware of the occurrence of interstitial disease in a more diffused bases that can occur from inorganic dust." (TX at 125). Drs. Wheeler and Scott, however, have greater credentials relative to reading CT Scans than Dr. James does. Therefore, I must credit both of their negative readings of the February 9, 1998 CT-Scan. Although it remains possible for Claimant to establish pneumoconiosis through other means, she has failed to do so by the CT-Scan evidence of record.

b. Biopsy or Autopsy Evidence and Medical Opinion Evidence

For purposes of this decision, the autopsy and medical opinion evidence will be addressed together. Pursuant to §718.202(a)(2), a claimant may establish the existence of pneumoconiosis by biopsy or autopsy evidence. Autopsy evidence is the most reliable evidence of the existence of pneumoconiosis. *Terlip v. Director, OWCP,* 8 B.L.R. 1-363 (1985). Autopsy reports must be given significant probative value regarding the existence and degree of pneumoconiosis because the pathologist who performs the autopsy sees the entire respiratory system as well as other body systems. *Fetterman v. Director, OWCP,* 7 B.L.R. 1-688, 1-691 (1985). In addition, it is reasonable to assign greater weight to the opinion of the physician who performs the autopsy over the opinions of others who review his or her findings. *Northern Coal Co., v. Director, OWCP,* 100 F.3d 871, 874 (10th Cir. 1996). However, a death certificate, in and of itself, is an unreliable report of the miner's condition. *Smith v. Camco Mining, Inc.,* 13 B.L.R 1017 (1989); *Addison v. OWCP,* 11 B.L.R 1-68 (1988).

Moreover, if scientific, technical, or other specialized knowledge will assist the judge as trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise. 29 C.F.R. § 18.702. A theory or technique may be said to be scientific knowledge that will assist the trier of fact if "it can (and has been) tested. *Daubert v. Merrell DowPharmaceuticals, Inc.*, 509 U.S. 579, 593 (1993). An additional consideration is whether the theory or technique has been "subjected to peer review and publication." *Id.* "*Daubert* does not apply directly in black lung cases, because it is based on Fed.R.Evid. 702, which agencies need not follow." *Peabody Coal Company v. McCandless*, ___ F.3d ___ (7th Cir. 2001), 2001 WL 726609 (7th Cir), 95-3291, 00-1449 & 00-2788. However, *Daubert* has been relied upon in black lung cases. *See id.*

The existence of pneumoconiosis may also be established by medical opinion evidence under §718.202 as set forth in subparagraph (a)(4).

> A determination of the existence of pneumoconiosis may be made, notwithstanding a negative x-ray, if a physician, exercising

> sound medical judgment finds the miner suffers or suffered from pneumoconiosis as defined in §718.201. Any such finding shall be based on objective medical evidence, such as arterial blood gas studies, pulmonary function studies, physical examinations, and medical and work histories. Such a finding must be supported by a reasoned medical opinion.

§718.202(a)(4).

A "reasoned" medical opinion is one in which the judge determines that the underlying documentation and data are adequate to support the physician's conclusions. *Fields v. Island Creek Coal Co.,* 10 B.L.R. 1-19 (1997). An unreasoned or undocumented opinion may be given little of no weight. *Clark v. Karst-Robbins Coal Co.,* 12 B.L.R. 1-149 (1989) (en banc). Moreover, an unsupported medical conclusion is not a reasoned diagnosis. *Fuller v. Gibralter Corp.,* 6 B.L.R. 1-1292 (1984).

In addition, it is permissible for an Administrative Law Judge (ALJ) to give more weight to physicians he finds have superior qualifications or those who perform more thorough medical evaluations of the miner's condition. *See Northern Coal Co.,* 100 F.3d at 874 (citing *Hansen v. Director, OWCP,* 984 F.2d 364, 368 (10th Cir. 1993). The ALJ, however, must provide a reasoned explanation of his preference of one physician over another. *See Northern Coal Co.,* 100 F.3d at 876 (citing *Hobbs v. Clinchfield Coal Co.,* 45 F.3d 819, 820 (4th Cir. 1995). In addition, the ALJ may consider the relationship between the patient and the treating physician. *Frey V. Bowen,* 816 F.2d 508 (10th Cir. 1987).

Autopsy Report

Todd L. Randolph, M.D., a staff pathologist at LDS Hospital, performed the autopsy on June 8, 1998. (DX 6). The slides from this autopsy are the basis for all the autopsy evaluations in the record. Dr. Randolph's final anatomical diagnosis was a clinical history of pulmonary fibrosis and increasing hypoxia with diffuse alveolar damage and pulmonary fibrosis. He did not report the cause of Mr. Jones' death. The autopsy was limited to the serous cavities, heart, vascular system, lungs, abdominal organs, pancreas, gastrointestinal system, spleen, and urinary tract.

The clinical history contained in the autopsy report indicated that Mr. Jones was a 75 year old male with a history of pulmonary fibrosis, hypoxia, COPD and oxygen dependence for two years. Recently prior to his death, he experienced an increase in shortness of breath and cough and was admitted to the hospital where he became increasingly hypoxic with complicated deep venous thrombosis. He expired on June 6, 1998. (DX 6 at 2).

Dr. Randolph's description of the lungs indicated the following:

> Right lung- 950 grams, left lung- 800 grams. There is no atelectasis. The trachea is complete. There is no evidence of aspirated material. The distal bronchi contain scant mucus. The pleural surfaces are smooth and glistening. The lungs and hilar nodes are severely anthracotic, and there is mild evidence of emphysema. On cut section, there is aspirated blood apparent in the alveoli. Bronchopneumonia is not recognized. There is no focal consolidation and no tumor. There is mild passive congestion of the lungs. There is evidence of pulmonary edema. The lungs are diffusely firm and lack crepitus. Pulmonary thromboemboli are not identified.

(DX 6 at 3-4).

Dr. Randolph's description of the heart indicated the following:

> The 430 gram heart is in usual position. The left ventricle is firm. The left ventricle is slightly hypertrophied and the cardiac chambers are not dilated. The right ventricle is not significantly hypertrophied. On opening the aorta and pulmonary trunk, there is no evidence of pulmonary thromboembolism. The circumflex coronary artery arises from the left main coronary artery. The coronary arteries are examined by multiple cross-sections. There is minimal calcific atherosclerosis in the main coronary arteries.
>
> Thrombosis of the coronary artery is not present. The cardiac valve leaflets are primarily delicate, translucent membranous. There is focal calcification. Vegetations are not identified.
>
> There is no softening or mottling of the myocardium. There is no significant myocardial fibrosis.

(DX 6 at 3).

Dr. Randolph, the prosector, had the best opportunity to examine Mr. Jones' lungs. I credit these finding because Dr. Randolph viewed Mr. Jones' lungs first hand and because there is an abundance of evidence showing that Mr. Jones had end stage pulmonary disease.

Dr. Green's Review of the Autopsy

At the request of Claimant, Francis H.Y. Green, M.B., Ch.B, M.D., a pathologist, submitted a report dated September 27, 2000[4]. (CX 1). Dr. Green is a Diplomate of the American Board of Pathology. In addition, he is a professor in the Department of Pathology and a co-chair of the Respiratory Research Group at the Faculty of Medicine at the University of Calgary. Dr. Green is the Head of Autopsy Service at Foothills Hospital in Calgary. He has also published extensively in his field. In preparation of his report, Dr. Green reviewed Mr. Jones' death certificate, Dr. Todd's autopsy report, seventeen (17) autopsy slides, medical records from Columbia Castleview Hospital and LDS Hospital, medical reports from Drs. Naeye, Perper, and Farney, chest x-rays, and the results of a pulmonary function test administered on May 27, 1987.

Dr. Green's report summarizes Mr. Jones occupational history, smoking history, and clinical history. He opined that Mr. Jones' lung disease became clinically apparent and required supplemental oxygen for treatment approximately two to three years prior to death, but that a review of the medical record shows that there was "radiographic, functional and blood gas evidence for a restrictive lung disease many years prior to his disease becoming clinically apparent." (CX 1 at 4). Dr. Green based his opinion on the following: (1) Mr. Jones' 1986 and 1997 blood gas studies revealed hypoxemia and met the Department of Labor's standard for disability; (2) there was a progressive decline between 1987 and 1998 in the forced vital capacity (FVC) which is a good index of restrictive lung disease; (3) medical examinations in 1986 and 1987 noted dyspnea which was possibly due to restrictive lung disease; (4) chest x-rays from 1982 noted hilar calcification which is a sign of silica exposure; and (5) as early as 1995 there were irregular markings noted at the basis of the lung on x-rays. Dr. Green opined that the only other medical history of significance was the presence of hypertension and gout.

Dr. Green also reviewed the autopsy slides. Based on his review, he concluded:

> The lungs showed a very severe interstitial fibrosis involving both upper and lower lung zones . . .. The pattern of pulmonary fibrosis histologically does not fit that of usual interstitial pneumonia (UIP) or idiopathic pulmonary fibrosis (IPF) in that in those conditions the fibrosis is of varying ages with advanced fibrosis adjacent to relatively normal areas of lung. These features were not present in Mr. Jones' lungs, and therefore from a histological point of view one can rule out the diagnosis of UIP or IPF. Furthermore, interstitial pulmonary fibrosis is a well-recognized feature of coal workers' pneumoconiosis, both pathologically and based on

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[4] Dr. Green's curriculum vitae is contained in the record at CX 2.

> radiologic studies of coal miners[5]. . .. It is noteworthy in this regard that the ILO classification system for the pneumoconiosis specifically recognizes the presence of irregular opacities as a component of coal workers' pneumoconiosis. The pathologic equivalent of irregular opacities on the radiograph is the presence of interstitial fibrosis. A very important diagnostic feature that was overlooked or misdiagnosed in previous pathologic reports was the presence of alveolar lipoproteinosis. This is a characteristic feature of the rapidly progressive form of silicosis.

(CX 1 at 4)

Dr. Green also reports that there was black pigment present in Mr. Jones' lungs. He used both a low power and a high power microscope to examine the pigment. His examination revealed the following:

> On low power examination, black pigment is seen throughout the lung tissue, but is most pronounced around small airways and blood vessels. In these areas, the remnants of dust macules can be seen. The amount of dust in the lungs is heavy and extends out into the areas of interstitial fibrosis. At higher magnification, many of the black particles are consistent with coal mine dust and, by polarizing microscopy, large numbers of birefringent particles are seen. Many of these have an acicular (needle) like shape: others are square and triangular. The former are characteristic of silicate minerals of various kinds; the latter are characteristic of silica (quartz) particles. No distinct silicotic nodules are seen, but in some areas there are micronodules.

(CX 1 at 3).

Dr. Green opined that "[t]he presence of large quantities of mineral dusts, including coal, in Mr. Jones' lungs is consistent with his occupational history." (CX 1 at 5). He noted that Mr. Jones worked in the mines as a motor man, shuttle car operator, general laborer, and lamp man. Dr. Green refers to a study[6] of U.S. coal miners which he opined showed a "relatively high prevalence (13%) of U.S. coal miners at death and that the risk for developing

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[5] Dr. Green supplied a list of scientific publications that support this assertion. *See* CX 1 at appendix 1.

[6] *Am J Indust Med* 16:605-615 (1989).

silicosis was greatest in miners who worked in certain jobs." (CX 1 at 6). "Among these high risk jobs were motor men and shuttle car operators who are exposed to considerable silica from pulverized dust on the rail car tracks." (CX 1 at 6).

Dr. Green concluded that Mr. Jones' lungs showed he had mixed dust pneumoconiosis which is a variant of pneumoconiosis, alveolar proteinosis consistent with rapidly progressive silicosis, and pulmonary vascular changes, consistent with pulmonary hypertension and cor pulmonale. Dr. Green acknowledged that "[a] slightly unusual feature of Mr. Jones' pneumoconiosis is its relatively late appearance as a severe progressive interstitial lung disease." (CX 1 at 5). He continued, "[a]lthough simple CWP rarely progresses in the absence of further exposure (and in this respect I agree with the opinion of Dr. Naeye), this is not the case in individuals exposed to high quantities of silica dust." (CX 1 at 5). "Silicosis may manifest after a post-exposure interval of many years[7]." (CX 1 at 5).

Dr. Green is a pathologist with exceptional credentials. His report is detailed, well reasoned, and provides an in-depth review of Mr. Jones' occupational and clinical history as well as the results of the autopsy. He found that Mr. Jones suffered from severe interstitial fibrosis, which is a feature of mixed dust fibrosis and of silicosis. However, Dr. Green also explained that Mr. Jones' interstitial fibrosis was not usual or idiopathic because the fibrosis was not of varying ages with advanced fibrosis adjacent to relatively normal areas of lung as is the case with those forms of fibrosis. In addition, he cited several studies in support of his position that interstitial pulmonary fibrosis is a well recognized feature of coal workers' pneumoconiosis. These studies have been published, tested, and subjected to peer review. Accordingly, I credit these studies and I find they have assisted the undersigned as the trier of fact in reaching a decision in this case.

Unlike the other physicians who examined the autopsy slides, Dr. Green used a high powered microscope during his examination of the slides. This high powered examination revealed anthrocotic and mineral dust particles in the lungs. In addition, his examination of the slides revealed black pigment and dust particles that appeared to be composed of a mixture of silicates and silica which Dr. Green argued were consistent with Mr. Jones' coal mine work history. Moreover, Dr. Green acknowledged that Mr. Jones' symptoms did not manifest until more than ten years after he left his last job in the coal mine. However, he also explained that a delay is common in individuals such as Mr. Jones who are exposed to high quantities of silica dust. Based on the detail and thoroughness of his well reasoned report, as well as his excellent credentials, I credit Dr. Green's review of Mr. Jones' autopsy records and his opinion that Mr. Jones died as a result of progressive interstitial lung disease caused entirely

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[7] Seaton A. Silicosis. In: Morgan WKC, Seaton A. eds. *Occupational Lung Diseases,* Philadelphia, Saunders, 1995, pp. 222-267; Symansky H. Delayed onset of sandstone pneumoconiosis. *Amer J Med* 2:101-102, 1981.

by exposure to dust within the coal mines which contained a high concentration of silicates and silica.

Dr. Perper's Review of the Autopsy

At the request of the District Director, Joshua Perper, M.D., LL.B., M.Sc., submitted a report dated February 25, 1999. (DX 7). Dr. Perper is a forensic pathologist and medical-legal consultant[8]. In addition to his medical degree, Dr. Perper also holds a law degree and a masters degree in forensic pathology. He is Board Certified in anatomical pathology, surgical pathology, and forensic pathology and is a Clinical Professor of pathology, epidemiology, and public health at the University of Miami. He is extensively published in his fields.

In preparation of his report, Dr. Perper reviewed the death certificate, occupational history, autopsy report dated June 8, 1998, eight (8) autopsy slides, and LDS Hospital records from May 11, 1998 through June 6, 1998. In addition, his report outlines Mr. Jones' occupational history, clinical history, and his lack of a smoking history.

Dr. Perper observed the following microscopic findings of Mr. Jones' lungs:

> Marked distortion of the normal pulmonary architecture, due primarily to an interstitial pathological process and to destructive alveolar changes. Slight focal thickening and fibrosis of pleura, with slight anthracotic pigmentation and a few birefringent silica crystals . . .. The anthracotic pigmentation is slight to moderate, focal, with presence of small to moderate numbers of silica crystals, and is seen primarily around blood vessels and in some of the inter-alveolar septa. Anthrocotic pigmentation within macrophages in the interstitium. . .. In a few places the thickened septa, expand into wider areas of compact fibrosis with similar changes. In places both the bronchi and the honeycomb spaces are filled with desquamated cells. . .. In places the dilated alveolar spaces contain small aggregates of macrophages laden with anthracotic pigment.

(DX 7 at 5-6).

Based on his review of the autopsy evidence, Dr. Perper made the following microscopic diagnoses:

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Dr. Perper's curriculum vitae is contained in the record at DX 8.

> Interstitial Pulmonary Fibrosis Variant of Coal workers' Pneumoconiosis (Diffuse and anthrocotic pigmentation, slight to moderate with silica crystals and associated emphysematous changes). Acute bronchopneumonia. Focal intra-alveolar hemorrhages.

(DX 7 at 6).

In addition, Dr. Perper based his finding of evidence of coal workers' pneumoconiosis on the following factors: (1) sufficiently long occupational exposure to coal dust to contract coal workers' pneumoconiosis or other occupational dust disease, (2) clinical records indicating Mr. Jones suffered from shortness of breath on exertion, cough productive of mucus, marked wheezing and required respiratory medications, steroids, and oxygen therapy in the years prior to death, (3) respiratory functions which showed hypoxemia, (4) despite a diagnosis of hypertension, no clinical diagnosis of heart disease or primary cardiac disease was made, the heart was not enlarged, and there was no evidence of coronary artery disease or myocardial damage, (5) Mr. Jones was not a smoker, and (5) chest x-rays read during life as showing pulmonary fibrosis with honeycombing and being negative for pneumoconiosis and re-readings after death which indicated the presence of severe nodular pneumoconiosis.

Dr. Perper's report states that the pathological findings at autopsy "did not substantiate the presence of typical maculo-nodular coal workers' pneumoconiosis, but disclosed the presence of an unusual pattern of severe interstitial fibrosis which the prosector labeled as idiopathic." (DX 7 at 7). He goes on to explain that:

> the prosector missed altogether the presence of silica crystals in the areas of interstitial fibrosis, and apparently was not familiar with the interstitial pattern of coal workers' pneumoconiosis. One cannot label a process of pulmonary fibrosis as idiopathic in the presence of significant occupational exposure to a fibrogenic dust such as coal dust containing silica, in particular when evidence of the silica is evident within the pulmonary fibrosis.

(DX 7 at 7).

Dr. Perper also found that the clinical and autopsy findings substantiated the fact that he had evidence of severe chronic pulmonary disease related to his occupational exposure as a miner. "The gross autopsy findings of severe diffuse interstitial pulmonary fibrosis, with marked honeycombing, and bullae, correlate well with the clinical findings of respiratory dysfunction and hypoxemia." (DX 7 at 7). Moreover, Dr. Perper cited several studies that substantiated the presence of interstitial diffuse pulmonary fibrosis in workers having coal miners' pneumoconiosis. "The scientific literature has substantiated that Diffuse Interstitial Fibrosis . . . of the lungs may be associated with non-asbestos pneumoconiosis including silicosis and mixed dust pneumoconiosis." (DX 7 at 8).

The first study cited by Dr. Perper is a 1993 Japanese study published in the *Respiration* journal entitled *Diffuse Interstitial Fibrosis in Non-asbestos Pneumoconiosis - A Pathological Study*. K. Honma & C. Keizo, *Diffuse Interstitial Fibrosis in Non-asbestos Pneumoconiosis - A Pathological Study*, 6 *Respiration*120-126 (1993). This study was performed on 233 autopsy cases with non-asbestos pneumoconiosis. Of this sample, 27.5 percent, or 64 cases, revealed evidence of Diffuse Interstitial Fibrosis (DIF). *Id.* In addition 3.9 percent, or 9 cases, had extensive disease resembling Usual Interstitial Pneumonia (idiopathic interstitial fibrosis). *Id.* "The study reported that there was no correlation between the occurrence of DIF and the type of the underlying disease (silicosis or mixed dust pneumoconiosis) except that an extensive DIF was more frequently associated with mixed dust pneumoconiosis.'" *Id.;* (DX 7 at 8).

Dr. Perper reported that similar findings were found in a 1997 French study entitled *Primary Diffuse Interstitial Fibrosis in Coal Miners; a NewEntity*. A. Brichet & B. Wallaert & B. Gosselin, *Primary Diffuse Interstitial Fibrosis in Coal Miners; a New Entity*, 14(4) *Rev Mal Resp* pp. 227-285 (Sept. 1997). This study involved 26 coal miners with a work history of twenty or more years with clinical, biological, and functional characteristics mimicking idiopathic pulmonary fibrosis (IDP). As Dr. Perper explained, "[a]ll had dyspnea and diffuse crackles. Eleven had finger clubbing. Computed tomography showed honeycombing (23 cases), ground opacities (6 cases) and bronchiectasis (3 cases). Nineteen (19) had radiological findings of CWP." (DX 7 at 8).

After reviewing all of this information, Dr. Perper concluded that in his professional opinion, within a reasonable degree of medical certainty, Mr. Jones had clinical evidence of severe chronic pulmonary disease which proved at autopsy to be a variant of CWP with diffuse and severe interstitial pulmonary fibrosis and honeycombing, that his CWP was a result of his long standing occupational exposure as a coal mine worker to mixed dust containing silica, and that CWP was the cause of death of Mr. Jones. (DX 7 at 9).

Dr. Perper's report is detailed, well reasoned, and provides an in-depth review of Mr. Jones' occupational and clinical history as well as the results of the autopsy. Dr. Perper concluded that Mr. Jones suffered from an interstitial pulmonary fibrosis variant of coal workers' pneumoconiosis. In addition, he opined that the prosector mislabeled Mr. Jones' fibrosis as idiopathic and that he could not have suffered from idiopathic fibrosis given the amount of dust he was exposed to while working in the mines. He found anthracotic pigmentation and a small to moderate number of silica crystals which the prosector did not find during the autopsy.

Dr. Perper also cited several studies in support of his argument that interstitial diffuse pulmonary fibrosis may be present in workers with non-asbestos pneumoconiosis including silicosis and mixed dust pneumoconiosis. Dr. Green and Dr. James agreed with the studies cited by Dr. Perper. Dr. James concentrates his work in the area of coal workers pneumoconiosis and he testified that these studies are published in a peer review and are

presented to the reviewers before the journal is published. (TX at 99). In addition, while Dr. Kleinerman did not expressly agree with Drs. Perper and Green regarding whether coal mine dust can modify interstitial fibrosis, he did state that it is "yet undetermined if the course and severity of the interstitial fibrosis is modified by coal mine dust." These studies have been published, tested, and subjected to peer review. Accordingly, I credit these studies and I find they have assisted the undersigned as the trier of fact in reaching a decision in this case that Mr. Jones suffered from coal workers pneumoconiosis. Accordingly, I find Dr. Perper's opinion thorough, detailed, and well reasoned and I credit this report.

Dr. James' Testimony and Medical Report Dated October 3, 2000

Dr. David S. James is a pulmonologist and is Board Certified in Internal Medicine, Pulmonary Diseases, and Critical Care Medicine[9]. (TX at 69-70). He is also a "B" reader and an Associate Professor of Medicine at the University of New Mexico School of Medicine. (TX at 70). Dr. James is the Medical Director of the Black Lung Clinic at the Miner's Colfax Medical Center in Ratone, New Mexico. (TX at 71). As Medical Director of the Black Lung Clinic, Dr. James' activities include overseeing respiratory therapists and a state-wide screening program that travels primarily in New Mexico and southern Colorado to provide health screening to miners for respiratory and other occupational illnesses. (TX at 71). The Black Lung Clinic receives federal funding and one of its purposes is to assist miners in obtaining black lung benefits. (TX at 109). Over the last eleven years, Dr. James has treated approximately 500 miners for respiratory disease. (TX at 69-70). In addition, he testifies frequently for claimants and has never testified for an employer. (TX at 110). Employer accepted Dr. James' credentials as a pulmonary expert. (TX at 70).

The following objective testing evidence is contained in the record and was evaluated by Dr. James in order to form his opinion.

Pulmonary Function Study Values

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[9] In addition to his testimony regarding his credentials, Dr. James' curriculum vitae is marked in the record as CX 4.

Ex. No.	Date	Age/Ht.	FEV1	FVC	MVV	FEV1/FVC
DX 32		64/70	2.47		62	
DX 32			Pre: 2.87 Post: 3.17	Pre: 4.06 Post: 4.00	Pre: 80 Post: 93	
DX 32		65/67.5	Pre: 2.87 (87%) Post: 3.17	Pre: 4.06 (96%) Post: 4.00	Pre: 80 (64%) Post: 93	
DX 21		65/67.5	Pre: 3.41 (98%) Post: 3.39	Pre: 4.04 (90%) Post: 4.04	Pre: 98 Post: 106	84
DX 20			2.01	3.39		
DX 20			2.48	2.81		
DX 21		74/70	Pre: 2.86 (85%) Post: 2.75	Pre: 3.41 (77%) Post: 3.42	Pre: 113 (92%) Post: 99	84
DX 16		74/70	Pre: 2.86 (85%) Post: 2.75	Pre: 3.41 (77%) Post: 3.42	Pre: 113 (92%) Post: 99	84

Arterial Blood Gas Study Values

EX. No.	Date	Physician	PCO2	PO2	Qualify
DX 32	11/12/86	Dr. Kotardy	36.6	57.5	Yes (58)
DX 32	01/05/87	Dr. Kennedy	37	56	DOL validation of 11/12/86 ABG
DX 32	10/07/87		35	57	Yes (60)
DX 21 and 16	07/16/96	Dr. Nichols	42.4	49.9	Yes (55)

Dr. James testified as follows on behalf of the Claimant at the hearing on October 25, 2000 (TX at 68-135). He issued a medical report dated October 3, 2000 which made three

cardiopulmonary diagnoses: (1) diffused interstitial fibrosis, (2) cor pulmonalia due to diffused interstitial fibrosis, and (3) deep venus thrombosis. (TX at 32, 76; CX 3). Dr. James is not a pathologist. (TX at 110). In order to prepare his report, he reviewed Director's exhibits 1-34 and the medical review and opinion of Frances Green, M.D., a pulmonary pathologist, dated September 27, 2000. (TX at 72-73; CX 3). Dr. James also reviewed and interpreted chest x-rays taken of Mr. Jones dated May 11, 14, 27, and 28, 1998, and June 6, 1998, as well as a CT scan of Mr. Jones' chest dated May 9, 1998. (CX 3). He performed a "B" reading of Mr. Jones' May 27, 1998 x-ray. (TX at 72). Dr. James never performed a clinical evaluation of Mr. Jones. (TX at 73; CX 3). In addition, although he has had the occasion to review autopsy slides in the course of his practice, at the time of the October 25, 2000 hearing, he had not reviewed Mr. Jones' autopsy slides. (TX at 74).

Dr. James' October 3, 2000 report contained a detailed summary of Mr. Jones' occupational and exposure history. He noted that Mr. Jones hauled and delivered coal from 1939 until 1943 when he entered the army until 1946. He also noted that Mr. Jones returned to coal mine employment where he worked as a motor man, shuttle car operator, laborer, and lampman from 1946 until his retirement in 1985. In writing about Mr. Jones' occupational and exposure history, Dr. James cited an office note by James Pearl, M.D. dated February 9, 1998 which states that Mr. Jones did not wear breathing protection as a miner. (CX 3).

Dr. James' October 3, 2000 report also contained a detailed summary of Mr. Jones' medical and smoking history. Based on Mr. Jones' medical examination by Dr. Lincoln in November 1986, Dr. James concluded he had smoked a pack of cigarettes a day until 1955 for a total of four to five pack years. Dr. James agreed with Dr. Lincoln's 1986 medical opinion and Dr. Farney's 1987 medical opinion and concluded that at that time period, Mr. Jones' condition was not related to his exposure to coal mine dust, he did not have pneumoconiosis, and there was no evidence of a respiratory impairment which would have limited Mr. Jones from performing his last job as a bathhouse attendant. (TX at 119-121). He also stated that in hindsight, the borderline abnormalities that were present in Mr. Jones at that time "may have been representative of Mr. Jones' ultimate diagnosis of interstitial fibrosis." (TX at 120-121).

Dr. James' first diagnosis was diffused interstitial fibrosis. (TX at 76, 84; CX 3). He based this diagnosis on clinical, radiographic, and pathologic evidence. (TX at 76). Mr. Jones' lungs showed honeycombing. Dr. James explained that "[h]oneycombing is an advanced development of interstitial lung disease of a variety of causes and it is almost always associated with just severe respiratory impairment." (TX at 108). In addition, Dr. James testified that the May 27, 1998 film he performed a B reading of on October 3, 2000 was representative of the other films of Mr. Jones' chest. (TX at 76). He interpreted this x-ray as showing parenchymal abnormalities consistent with pneumoconiosis. (TX at 77).

Dr. James acknowledged that there are several B readings obtained by Employer from Dr. Wheeler and Dr. Scott that did not indicate parenchymal abnormalities consistent with pneumoconiosis. (TX at 79; EX 28). Dr. James quoted from Dr. Wheeler's B reading on a film from May 28, 1998 which stated:

> Minimal to moderate non-specific interstitial fibrosis, edma or infiltrate in lower lung zones and lateral portion of both mid-lungs compatible with usual interstitial pneumonitis or CHF. Suggest clinical correlation. Hypoinflation of lungs with minimal right diaphragmatic elevation. Obesity.

(TR at 80-81; EX 28). Based on these comments, Dr. James concluded that Dr. Wheeler found interstitial fibrosis. He further explained that parenchymal abnormalities consistent with pneumoconiosis is a form of interstitial fibrosis. (TX at 81).

> What I'm trying to get across is Dr. Wheeler and Dr. Scott it is their opinion there is no pneumoconiosis based on the findings that they're seeing on the x-ray. They are findings - abnormal findings - on the radiograph. It's their opinion that they're not due to pneumoconiosis. The same abnormalities that are on the radiograph are the same ones that I'm calling consistent with pneumoconiosis.

(TX at 127). However, Dr. James also opined that "you cannot determine on the radiograph whether it's usual interstitial pneumonitis or other cause of pulmonary fibrosis with certainty." (TX at 84).

In addition, Dr. James testified that there are different autoimmune or collagen vascular diseases that are associated with fibrosis and that it is possible there could be remnants of the atypical pneumonia Mr. Jones was diagnosed with in 1995 that are visible on the x-ray he viewed. (TX at 112-114). Atypical pneumonia is not bacterial, but can represent pneumonia from other agents, such as fungi, parasites, and viruses. (TX at 113). On cross examination, Dr. James was referred to a report dating from May 10,1982 to May 15, 1982 which also makes a reference to atypical pneumonia. (TX at 114). He said that the 1995 atypical pneumonia was less likely to be a continuation or reappearance of the 1982 atypical pneumonia described in the 1982 report. (TX at 115). However, there are infectious pneumonia that can leave changes in the x-ray after the clinical course has gone back to normal. (TX at 115-116). He also said that it was unlikely histoplasmosis, a fungus that is endemic in certain parts of the country and can cause changes on an x-ray, caused the changes on the autopsy slides. (TX at 116).

Dr. James also opined that idiopathic pulmonary fibrosis does not explain the clinical condition or cause of Mr. Jones' pulmonary fibrosis. (TX at 81). He based this opinion on the following:

> Idiopathic pulmonary fibrosis is used by myself and I think by many pulmonologists as a fibrotic condition where there is no known cause of the fibrosis as the name implies, idiopathic. So if there is what is felt to be a causative agent then it's usually not

> called idiopathic pulmonary fibrosis. So, in general, there are a number of different type of fibrotic lung diseases. The idiopathic pulmonary fibrosis often has a clinical picture, but you need to exclude other causes of that pattern if you can and I feel there are some other likely causes of the fibrosis in Mr. Jones' case. Part of that is - or an important part is looking at the actual pathology that, in addition to the fibrosis, Dr. Perper and Dr. Green also saw other findings that would suggest that it's not idiopathic. Once, they found evidence of dust deposited and specifically what was felt to be silica dust deposited in the lung tissue. Dr. Green also felt there was a condition called alveolar proteinosis, which is one condition that can occur from heavy silica exposure and it's a response to heavy silica exposure and that was in Dr. Green's narrative, an important finding and I think myself I also consider that important in that that is not a feature of other causes that on tissue level clinically could be considered idiopathic pulmonary fibrosis, such as usual interstitial pneumonitis. This alveolar proteinosis for one is not associated with that condition and in the setting of a dust exposed individual with dust in the fibrotic area of the tissue that is an important finding that is related to the development of the fibrosis.

(TX at 81-82).

Dr. James further testified as follows:

Mr. Wilderman: In other words, the development of the fibrosis is related to the presence of the silica particle which in turn is part of the coal mine dust. Is that what you're saying?

Dr. James: That's part of it. And the development of fibrosis is due to silica exposure as well.

Mr. Wilderman: So that by identifying silica particles in the pattern or form in which it was reported by the pathologist and the presence of the alveolar proteinosis, do you agree, based on that or the opinion of Dr. Green, that that excludes IPF as a diagnosis?

Dr. James: I feel it does, yes.

(TX at 82-83).

Dr. James opined that Mr. Jones did not have usual interstitial pneumonitis. This opinion was based on:

> the presence of the alveolar proteinosis is not a characteristic of usual interstitial pneumonitis. The presence of the dust and what appears to be silica dust within the fibrotic tissue would not be frequently seen in the setting of usual interstitial pneumonitis. So it's the two finding of the dust and the alveolar proteinosis, which make this less likely [usual interstitial pneumonitis] and more likely another fibrotic condition.

(TX at 85; DX 7).

Dr. James also testified that Mr. Jones had cor pulmonale. (TX at 85). Dr. James is familiar with the Black Lung regulations regarding cor pulmonale. (TX at 89). Cor pulmonale causes respiratory impairment. (TX at 93). Although he did not view the autopsy slides before the hearing, he viewed Dr. Green's report dated September 27, 2000 which indicated a diagnosis of cor pulmonale. (TX at 89). Dr. James stated that his diagnosis of cor pulmonale is supported by the following evidence:

> The main finding were that he had a respiratory condition, his diffused interstitial fibrosis, which I felt was severe enough to increase his likelihood of developing cor pulmonale and Dr. Green's analysis of the tissue that was evidenced in the pulmonary vessels that he felt were consistent with increased pressure cor pulmonale based on the findings in the pulmonary vessels.

(TX at 91).

Right heart failure is a form a congestive heart failure which would be a manifestation of cor pulmonale. (TX at 139). Dr. James testified that to a reasonable degree of medical certainty, it is more likely than not that cor pulmonale produced or contributed to Mr. Jones' respiratory impairment and that this cor pulmonale was related to Mr. Jones' dust exposure in coal mine employment. (TX at 93). It is related to his dust exposure "due to the interstitial fibrosis . . . which was severe enough to cause cor pulmonale and in turn, it's my [Dr. James'] opinion that that was a consequence of his dust exposure as a miner. (TX at 94). The original prosector, Dr. Todd Randolph, did not find evidence of cor pulmonale. (TX at 91, 111).

Dr. James also cited Dr. Perper's medical opinion which found that the prosector did not diagnose right ventricular hypertrophy consistent with cor pulmonale and also failed to report the thickness of the ventricular wall. (TX at 92). Accordingly, Dr. James opined that the autopsy prosecutor did not do enough. (TX at 92). Dr. James also referred to Dr. Perper's conclusion that the prosector missed altogether the presence of silica crystals in the area of interstitial fibrosis. (TX at 93). Dr. James opined that diffuse interstitial fibrosis and cor pulmonale are chronic lung diseases, and in this case were significantly aggravated by exposure in Mr. Jones coal mine work. (TX at 95).

Dr. James also testified that Mr. Jones' respiratory disease became worse over time. For example, the values of Mr. Jones' blood gas studies of November 12, 1986, October 7, 1987, July 16, 1996, and October 30, 1997 meet the Department of Labor standard for disability. (TX at 99-100). In addition, the values declined significantly, thus showing a progression of Mr. Jones' respiratory disease. (TX at 101).

Dr. James stated that pulmonary function studies do not indicate the cause of abnormality. (TX at 85). The most recent study was on March 9, 1998 and produced the following results: FVC- 2.81 liters which was 80% of normal, FEV-1- 2.48 liters which was 92% of normal. In February 9, 1998 the FVC was 2.39 liters which was 67% of predicted and the FEV-1 was 2.01 liters which is 74% of predicted. Dr. James also stated that there were arterial blood gas test results that meet the DOL Standard for disability going back to November 1986. In the November 1986 test, the Po2 was 57.5 and the Pco-2 was 36.6. In the October 30, 1997 test, the Pco-2 was 35 and the Po-2 was 57. These are qualifying values. (TX at 100). There was a blood gas test done on July 16, 1996, At that test, the po-2 was 49.9 and the Pco-2 was 42.4. These are qualifying values. Dr. James concluded that these values demonstrate a progression of Mr. Jones respiratory disease. He said it was his opinion that coal mine dust related disease can progress after cessation of exposure. Dr. James also testified that "[i]ndividuals with more severe lung disease, in general, there would be a progressive respiratory failure that the respiratory system cannot maintain gas exchange, oxygen levels." (TX at 106).

Dr. James concluded that cor pulmonale played a contributing factor that hastened his death and had Mr. Jones not had cor pulmonale, his would not have died when he did. (TX at 106). Dr. James opined with medical certainty that the cause of Mr. Jones' death was respiratory failure secondary to his underlying lung and heart disease. (TX at 107). Mr. Jones' respiratory failure was primarily caused by pneumoconiosis. (TX at 107).

Dr. James has excellent credentials and his report and testimony was thorough and well reasoned. He extensively considered Mr. Jones' medical, work, and smoking history and the effect they had on his health condition at death. In addition, he based his opinion on clinical, radiological, and pathological evidence as well as medical literature on the subject. I find that he adequately explained Mr. Jones' unusual form of pneumoconiosis that arose several years after he left his last coal mine employment. Accordingly, I credit the medical opinion of Dr. James that Mr. Jones' coal mine dust exposure contributed to the medical condition that caused his death.

At Claimant's request, David S. James, M.D. submitted an additional medical report dated February 11, 2001 that provided a review of the autopsy evidence. (CX 6).

Dr. James examination of the autopsy slides revealed the following:

> There were very few areas of more normal appearing lung tissue. The interstitial spaces were infiltrated with varying degrees of

> fibrosis . . .. There were regions in both lungs . . . showing areas of darkly pigmented material deposited in areas of fibrosis. I did not identify any silicotic nodules or coal macules in any of the lung slides. The slides were not viewed under a polarizing microscope.

(CX 6 at 1).

Dr. James also found that diffuse fibrosis was present in all of the lung sections from both lungs. In addition, he found there was a "lack of a significant number of polymorphonuclear cells in most of the lung sections." (CX 6 at 2). Dr. James explained that "[t]his decreases the probability that an acute infectious process shortly before Mr. Jones' demise was a causative factor in the development of his diffuse lung disease." (CX 6 at 2).

Dr. James is familiar with and agrees with the studies cited in Dr. Green's report (CX 1, Appendix 1) which support the Claimant's assertion that interstitial fibrosis is a feature of coal workers pneumoconiosis mixed with dust fibrosis and silicosis. (TX at 98). Dr. James opined that coal mine dust related respiratory disease can progress after cessation of exposure. (TX at 101). Dr. James cited an article from the *British Journal of Industrial Medicine,* 1983, Volume 40, page 169 for support of this opinion. (TX at 101-102). This article demonstrated that about 26% of miners had worse x-ray profusion after they ceased mining. TX 102. Dr. James said that there was some evidence that Mr. Jones' symptoms occurred even earlier than 1995 as opined by Dr. Naeye and that these symptoms were progressive. TX 103. However, Dr. James admitted that there were medical studies that did not agree with his linking of coal mine dust and interstitial fibrosis. TX 131-134. He stated that he had more confidence with studies that show progression in pneumoconiosis and it was his opinion that the form of coal workers' pneumoconiosis that Mr. Jones had as well as other forms can be progressive. (TX at 143). In addition, Dr. James believes that the studies cited by Drs. Green and Perper are regarded as reliable medical authority and are commonly relied upon by academicians and clinicians in the practice or in the field of pulmonary medicine. (TX at 98). Dr. James testified that they are accepted in the medical community.

Dr. James' report based on the pathological evidence is also detailed, well reasoned, and provides an in-depth review of Mr. Jones' occupational and clinical history as well as the results of the autopsy. In addition, Dr. James' review of the autopsy slides also revealed diffuse fibrosis in all lung sections and regions of dark pigment in some regions. He opined that the coal mine dust he inhaled as a coal miner was a contributing causative factor in the development of his diffuse lung disease. Because he did not use a polarizing microscope, Dr. James was unable to assess whether there was silica present in Mr. Jones' lungs. However, he did refer to his October 3, 2000 medical report as well as his testimony before the undersigned in which he stated that "coal miners are at risk for developing diffuse fibrotic lung disease even if coal macules or silicotic nodules are not present." In addition, Dr. James explained that the probability that an acute infectious process was a causative factor in the development of Mr. Jones' lung disease because there were an insufficient number of

polymorphonuclear cells present. Dr. James has excellent credentials and he concentrates his medical work in the area of black lung. Accordingly, I credit his detailed, thorough, and well reasoned opinion.

Dr. Naeye's Review of the Autopsy

At the request of the District Director, Richard L. Naeye, M.D. submitted a report dated June 21, 1999[10]. (DX 26). Dr. Naeye is Board Certified in anatomic and clinical pathology. In addition, he is a Professor of Pathology at the Pennsylvania State University College of Medicine as well as a member of the Editorial Board of Modern Pathology. He is also extensively published in the field of pathology.

In preparation of his report, Dr. Naeye reviewed Mr. Jones' death certificate, Dr. Todd's autopsy report, the medical records from LDS Hospital, Columbia Castleview Hospital, Dr. Perper's report, and reports from Drs. G.R. Goodman, D.A. Nichols, and several radiologists. Dr. Naeye examined eighteen (18) tissue slides, eight (8) of which were lung tissue slides, two (2) of which were heart tissue slides, and the rest which were slides of the other organs. In addition, Dr. Naeye considered Mr. Jones' occupational history, his medical history of hypertension and gout, a five pack year cigarette smoking history, as well as his clinical history. In addition, he noted the following:

> Two years before he died pulmonary function studies revealed an FEV_1 that was 85% of normal, and FVC that was 77% and an MVV that was 92%. These normal values did not increase after a bronchodilator was administered and they did not persist for long because he soon thereafter became progressively more dyspneic. X-rays consistently identified the presence of widespread interstitial fibrosis or fibrosis. None reported the presence of the upper lobe nodules which are characteristic findings of coal workers' pneumoconiosis.

(DX 26 at 1).

Dr. Naeye reported that a microscopic examination of the lung tissues revealed the following:

> [A] very severe interstitial fibrosis that has old consolidated fibrotic areas, very recent areas of subacute inflammation and necrosis and intermediate lesions where fibroblasts are active and collagen deposit is recent. . . . There is no more black

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10 Dr. Naeye's curriculum vitae is contained in the record at DX 26.

> pigment in this mans lungs than is present in the lungs of non-miners. The only evidence that this man ever mined coal is the presence of a few very tiny black deposits in the subpleural space or adjacent to small arteries and airways. . . . Also present in the lungs are some areas of edema and acute lumbar pneumonia.

(DX 26 at 1).

Based on his examination of the information provided for his review, Dr. Naeye opined that:

> There is not a trace of any lesion that could be coal worker's pneumoconiosis (CWP) in the lungs of this man. There are no anthracitic micro nodules present, larger black lesions or tiny birefringent crystals. Thus no lesions are present that fulfill the minimal criteria for the diagnosis of any form of coal worker's pneumoconiosis or silicosis. Death was due to a rapidly progressive interstitial fibrosis that over a period of two years destroyed this man's lungs. Acute lobar pneumonia had a minor role in his terminal hours. Being absent CWP did not cause an impairment in the lung function or hasten this man's death. He would have died at the same time and in the same way if he had never mined coal.

(DX 26 at 1-2).

Dr. Naeye disagreed with Dr. Perper's analysis of the autopsy for several reasons. First, Dr. Naeye stated that a diagnosis of CWP is made on the basis of the characteristic lesions it produces that are seen under the microscope and that none of the characteristic findings of CWP were present under the microscope in this autopsy. He also disagreed with Dr. Perper's diagnosis that the "respiratory functions showed hypoxemia" because hypoxemia is not a respiratory finding, it is a hematologic finding, and is most often the consequence of pulmonary insufficiency. Dr. Naeye reported that in this case, the miner has normal findings on pulmonary function tests just 2 years before he died, which was 13 years after his last coal mine employment. He also opined that "[o]ne of the characteristic features of simple pneumoconiosis is that it does not progress after a worker leaves the exposure to coal mine dust." (DX 26 at 2). He further remarked that "[t]here is no known type of CWP that causes diffuse interstitial fibrosis to first appear many years after retirement for the industry and then rapidly progress to destroy a man's lungs in two years." (DX 26 at 2).

At the request of Employer, Dr. Naeye submitted an additional report relative to the findings and interpretations of Drs. Perper, James, and Green dated October 5, 2000. (RX 3). In this report, Dr. Naeye disagreed with their findings and interpretations and concluded

that "the postulate of Drs. Green and James that the interstitial fibrosis in the lungs of Clell Jones had an origin in exposure to coal mine dust ten or more years before death is implausible." (RX 3 at 1). "This conclusion is supported by extensive literature on this subject[11]." (RX 3 at 1).

In his report, Dr. Naeye again stated "it has been repeatedly documented that simple CWP does not advance after a miner quits the industry." (RX 3 at 1). His report goes on to opine that:

> Therefore it is implausible to postulate that simple CWP first appeared and became symptomatic in 1995 or 1996. Examination of his lung tissues removed at autopsy reveal that the 1995-96 appearance of his pulmonary insufficiency was due to the onset and rapid progression of an interstitial disease process. This timing is incompatible with the development of any form of coal worker's pneumoconiosis (CWP) except complicated CWP. No one who has examined the lungs of Clell Jones has found the characteristic lesions of this latter disorder.

(RX 3 at 1).

Dr. Naeye also states that none of the studies cited by Dr. Perper to support his claim that interstitial lung disease can be caused by coal mine employment are based upon epidemiologic analyses and that there is no creditable evidence in the literature that diffuse interstitial fibrosis is more frequent in coal mines than in the general population.

Dr. Naeye also argues that the jobs Mr. Jones performed in the mine did not expose him to high levels of free silica such as working as a roof bolter or rock driller would. He goes on to explain:

> The marked discrepancy between the near normal $FEV_{1,}$ FVC values and this man's severe respiratory distress is explained by the fact that his lung damage was interstitial rather than located around small arteries, airways and in the subpleural space as is characteristic of coal worker's pneumoconiosis. Dr. James' reasoning that Clell Jones' interstitial lung disease originated in his exposure to coal mine dust is based on the proposition that such exposure can cause interstitial disease. This thesis cannot be supported by the facts. . .. The only thing in mine dust that might cause interstitial fibrosis is a very massive exposure to

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11 Dr. Naeye provided a list of studies to support his opinion. *See* RX 3 at 3.

> free silica. The interstitial disorder appears clinically between 6 months and 2 years after the exposure takes place. Clell Jones quit mining coal at least 10 years before signs and symptoms of his interstitial lung disease appeared.

(RX 3 at 2).

In both his 1999 and 2000 opinions, Dr. Naeye argues that Mr. Jones did not have pneumoconiosis because the disease does not progress after the worker leaves the coal mine dust. Dr. Naeye stated that there is extensive literature to back up this theory and in fact cites several of them. However, §718.201(c) of the regulations and the majority of the circuits, including the 10th circuit which has jurisdiction in this matter, have expressly recognized that pneumoconiosis is a latent and progressive disease. Therefore, despite Dr. Naeye's assertions to the contrary, I find that pneumoconiosis may progress once the miner has left his coal mine employment. Accordingly, I do not credit Dr. Naeye's opinion that Mr. Jones' pneumoconiosis could not have progressed after he left the coal mines. In addition, the reports cited by Drs. Green and Perper presented studies that show pneumoconiosis may appear following a miner's departure from his work. I have credited these studies. Although Dr. Naeye disagrees with the results of these studies and stated that "there is no creditable evidence in the literature that diffuse interstitial fibrosis is more frequent in coal mines than in the general population," he does not provide the basis for this opinion.

In addition, although he admits that silica dust causes pulmonary fibrosis, Dr. Naeye argued that Mr. Jones was not exposed to high levels of silica. However, Dr. Pearl, a treating physician, reported a history of coal dust exposure of significance and silica dust exposure. Dr. Naeye cites roof bolting and rock drilling as an example of a job in the mine that would expose a worker to silica and states that Mr. Jones did not perform any of these types of jobs. However, as Mr. Pierce testified, Mr. Jones did in fact work as a roof bolter. In addition, Mr. Jones' wife and son testified that Mr. Jones worked at the face where blasting and drilling occurred. Moreover, Dr. Green listed shuttle operator as another high risk job that exposes mine workers to dust. Mr. Jones spent the bulk of his time is the mines as a shuttle operator. Therefore, I find that Mr. Jones was in fact exposed to silica while working at the mines. It appears that Dr. Naeye either did not read Dr. Pearl's report or disregarded his report of Mr. Jones' dust exposure. Furthermore, through the use of a high powered microscope, Dr. Green discovered the presence of antracotic and mineral dust particles in the lungs as well as black dust particles. Dr. Naeye did not report having used a high powered microscope during his examination. Therefore, despite his excellent credentials, I do not find Dr. Naeye's reports to be thorough or well reasoned due to their failure to consider Dr. Pearl's stated history of Mr. Jones' significant coal and silica dust exposure.

Dr. Kleinerman's Review of the Autopsy

The Employer also submitted the medical report of Jerome Kleinerman, M.D [12]. dated July 31, 1999. (RX 1). Dr. Kleinerman was Board Certified in Pathologic Anatomy and Clinical Pathology. He began teaching in the area of pathology in 1951. At the time he wrote this report, Dr. Kleinerman was a Professor and the Vice Chairman of the Department of Pathology at Case Western Reserve School of Medicine, as well as the Director of the Department of Pathology at Metrohealth Medical Center in Cleveland, Ohio. He published extensively relative to pneumoconiosis and disease processes of the lung.

In preparation of his report, Dr. Kleinerman considered the autopsy report and slides, chest x-rays, medical records from Carbon Hospital, Castleview Hospital, and LDS Hospital, pulmonary function studies, arterial blood gas studies, and miscellaneous radiology and laboratory reports, medical reports and office notes from Drs. Nichols, Pearl, Naeye, and Perper, as well as Mr. Jones' death certificate.

In his report Dr. Kleinerman summarized Mr. Jones occupational history, smoking history, and clinical history. Regarding the pulmonary function studies on February 9, 1998 which revealed that the FVC and FEV-1 were mildly to moderately below predicted normal, Dr. Kleinerman opined that these values indicate the presence of mild to moderate extent of restrictive and obstructive lung dysfunction. Moreover, a March 9, 1998 pulmonary function study revealed that the FVC was slightly below normal and the FEV_1 was within normal limits. Dr. Kleinerman opined that this result indicated the presence of a mild degree of restrictive lung dysfunction but no evidence of obstructive lung dysfunction.

Dr. Kleinerman disagreed with Dr. Perper's opinion that Mr. Jones had a variant of CWP leading to interstitial pulmonary fibrosis and that pneumoconiosis was the cause of his death.

> All seven lung sections show extensive and severe nonspecific diffuse interstitial fibrosis with honeycombing . Honeycombing is a pattern of interstitial fibrosis in which the airspaces are enlarged and the surrounding airspace are enlarged and the surrounding airspace walls are thickened and fibrotic suggesting the appearance of a honeycomb. . .. A small amount of yellow brown to black pigment is present with the interstitial tissue. There are no lesions of simple nor of complicated coal workers' pneumoconiosis (CWP). The exudate within the airspaces in both the left and right lung is granular, hematoxic, and contains a small number of neutrophils indicating localized areas of early bronchopneumonia.

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Dr. Kleinerman's curriculum vitae is contained in RX 1. Unfortunately, a short time after this report was written, Dr. Kleinerman died as a result of an automobile accident.

(RX 1 at 6).

Dr. Kleinerman also stated that:

> There was a minimal amount of black granular pigment in the lung tissue but the pathologic evidence required for the diagnosis of simple CWP and/or for the diagnosis of complicated CWP as described in the *Archives of Pathology and Laboratory Medicine*, 103:375-432, July 1978, is not present in Mr. Jones' lung tissue. On this basis a diagnosis of simple CWP cannot and should not be made or accepted.

(RX 1 at 6).

Dr. Kleinerman disagreed with the studies Dr. Perper used to support his medical opinion that coal mine dust was the cause of the interstitial pulmonary fibrosis in Mr. Jones' lungs. He argued that the study entitled "Diffuse Interstitial Fibrosis in Nonasbestos Pneumoconiosis," was biased and was not a sample of the average working miners because it selected autopsied cases of the most severely ill patients and hospitalized patients who were moderately or severely ill. As a result, he opined the study produced a high indication of the incidence of interstitial fibrosis in the mining population. In addition, he stated the study did not attempt to "exclude other forms or other recognized causes of DIF such as collagen diseases, organizing pneumonia, drug induced pulmonary fibrosis or extrinsic allergic alveolitis with fibrosis." (RX 1 at 7). Therefore in his opinion this study provided "no scientifically reliable evidence that DIF occurs more frequently in a coal mining population than it does in a control population of non coal mining subjects." (RX 1 at 7).

Dr. Kleinerman also disagreed with the study entitled "Primary Diffuse Interstitial Fibrosis in Coal Miners: A New Entity." Dr. Kleinerman opined that the 412 miners in this study represented a highly selected group of hospitalized coal miners and that there was a limited attempt to exclude other known causes of interstitial pulmonary fibrosis. Moreover, Dr. Kleinerman disagreed with the third study cited by Dr. Perper in support of his conclusion that DIF may be caused by coal mine dust, *Interstitial Fibrosis in Coal Workers: Experience in Wales and West Virginia* published in *Ann Occup Hyg*, 32 (suppl l) 553-560, 1988 By McConnochie, Green, Vallyathan, Wagner, Seal and Lyons. He concluded that the authors consider "the presence of DIF a different and distinctive form of lung injury than that produced by coal mine dust." (RX 1 at 8). He goes on to state that "[i]t is as yet undetermined if the course and severity of the interstitial fibrosis is modified by coal mine dust." (RX 1 at 8).

Ultimately, Dr. Kleinerman opined:

> In conclusion it is my opinion with reasonable medical certainty that DIF is a distinctive and separate type of lung disease and is not caused by coal mine dust inhalation.

> I strongly disagree with Dr. Perper's conclusion that CWP and exposure to coal mine dust was a substantial cause of Mr. Jones' death. In my opinion and with reasonable medical certainty, Mr. Jones had no evidence of simple or complicated CWP in his lung tissues at autopsy. The amount of black granular pigment present in his lung tissue was minimal. There is therefore no pathologic evidence to support the belief the CWP or exposure to coal mine dust was a substantial cause of Mr. Jones' death. In my opinion and with reasonable medical certainty, Coal Workers' Pneumoconiosis and/or exposure to coal mine dust did not cause, did not contribute to, and did not hasten Mr. Jones' death.

(RX 1 at 8).

Through the use of a high powered microscope, Dr. Green discovered the presence of antracotic and mineral dust particles in the lungs as well as black dust particles. Dr. Kleinerman did not report having used a high powered microscope during his examination. Dr. Kleinerman failed to report that Mr. Jones was exposed to significant coal and silica dust as reported by Mr. Jones' treating physician, Dr. Pearl, who reported a history of coal dust exposure of significance and silica dust exposure. In addition, Claimant, Mr. Clell Jones, and Mr. Collier all testified that Mr. Jones was exposed to tremendous amounts of dust when he worked in the mines. It appears that Dr. Kleinerman either did not read Dr. Pearl's report or disregarded his report of Mr. Jones' dust exposure. Moreover, I do not find Dr. Kleinerman's rejection of the studies cited by Dr. Perper to be persuasive. Therefore, despite his excellent credentials, I do not find Dr. Kleinerman's report to be thorough, well reasoned, or a diagnosis of idiopathic pulmonary fibrosis due to his failure to consider Mr. Jones' coal and silica dust exposure and the fact that silica dust is a cause of interstitial fibrosis.

Dr. Tuteur's Review of the Autopsy

Employer offered the medical opinion of Peter G. Tuteur, M.D. dated November 9, 2000[13]. (EX 4). Dr. Tuteur is Board Certified in Internal Medicine and Pulmonary Disease. He is an Associate Professor of Medicine and the Director of the Pulmonary Function Laboratory at the Washington University School of Medicine in St. Louis, Missouri. He is also an associate physician at Barnes-Jewish Hospital in St. Louis.

Dr. Tuteur reviewed Mr. Jones' treatment records from Dr. Nichols, his hospital records from 1977 through Mr. Jones' death, his death certificate, the autopsy report prepared by Dr. Randolph, and the autopsy evaluations from Drs. Green, Perper, Naeye, and Kleinerman, as well as the independent review of Dr. James. Dr. Tuteur's report contains a summary of Mr.

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[13] Dr. Tuteur's curriculum vitae is marked in the record as RX 4.

Jones' medical history. He reported that in 1996, which he noted was ten years after he stopped working in the coal mine, Mr. Jones began to develop severe breathlessness at times associated with productive cough and rapidly progressive hypoxemia which eventually required supplemental oxygen. He also found radiograph evidence consistent with a progressive interstitial progress which was eventually diagnosed as idiopathic pulmonary fibrosis.

Dr. Tuteur reviewed the results of Mr. Jones' arterial blood gas studies and pulmonary function studies. He noted that by May 1998 there was an impairment of gas exchange with pO2 in the hypoxic level, which Dr. Tuteur opined was "consistent with impairment of gas exchange certainly due to the newly developed interstitial process." (EX 4 at 5). He went on to explain that "[t]hese data by virtue of the delay in the development of a restrictive abnormality to about ten years after the cessation of coal mine dust exposure reflect a high degree of unlikelihood that such changes were a result of a process caused by the inhalation of coal mine dust." (EX 4 at 5).

Dr. Tuteur also reviewed Mr. Jones' chest x-rays. He opined that:

> The almost ten year delay in conversion from normal radiographs to those consistent with a lower lung field interstitial process helps support the contention that the etiology of the interstitial process is not the inhalation of coal mine dust. To be sure, conversion of a negative to a positive radiograph may develop in persons with coal workers' pneumoconiosis, even after the cessation of coal mine dust exposure. When this does happen, it usually occurs in the first month or year following such dust exposure cessation. When this occurs, it typically results in an abnormality that is of relatively low profusion.

(EX 4 at 5-6).

Dr. Tuteur disagrees with both the French and Japanese studies cited by Drs. Perper and Green in support of the opinion that nonspecific interstitial fibrosis is found in coal miners with increased frequency because of their coal mine dust exposure. Dr. Tuteur found that while these studies show that diffuse interstitial fibrosis may occur in persons with environmental exposure, there was insufficient data in the studies to conclude that this frequency is greater than the general population. He also discredits the 1997 study entitled *Primary Diffuse Interstitial Fibrosis in Coal Miners; a New Entity* which found that diffuse interstitial fibrosis can occur in miners because he finds that its population was highly selected, which in turn limits any conclusion that implies that the frequency is greater than the general population.

Dr. Tuteur also discusses Dr. Green's opinion that the presence of silica crystals in the autopsy specimen was the result of Mr. Jones being exposed to silica. Dr. Tuteur, however, states that he agrees with Dr. Naeye who concluded that "rapidly progressive interstitial

processes of the lung can occur in response to silica exposure but do so only with extraordinarily intense and high concentration exposure such as occurs with unprotected sandblasting or tunnel drilling." (EX 4 at 6).

However, Dr. Tuteur does not discuss Mr. Jones' history of coal and silica dust exposure. Therefore, despite his excellent credentials, I do not credit Dr. Tuteur's opinion as thorough and well reasoned because he has not considered Mr. Jones' history of silica dust exposure as a cause of interstitial pulmonary fibrosis although he admits that silica dust causes pulmonary fibrosis. In addition, I do not find Dr. Tuteur's rejection of the studies cited by Drs. Perper and Green to be persuasive. He offers his opinion that the studies were based on insufficient data and a highly selected population, however, he does not provide the basis for this opinion. Accordingly, notwithstanding the excellent credentials of Drs. Naeye, Kleinerman, and Tuteur, I credit the opinions of Drs. Green, Perper, and James. Therefore, I also find that Claimant has established that Mr. Jones had pneumoconiosis through autopsy evidence.

C. Presumptions

Pursuant to §718.202(a)(3), a determination of the existence of pneumoconiosis may also be shown using the presumptions described in §§ 718.304, 718.305, and 718.306. Section 718.304 requires x-ray, biopsy, or equivalent evidence of complicated pneumoconiosis which is not present in this case. Section 718.305 applies only to claims filed before January 1, 1982. Because this case was filed after that date, the §718.305 presumption is inapplicable. Section 718.306 is only applicable in the case of a deceased miner who dies before March 1, 1978. Thus, the §718.306 presumption is not applicable either. Therefore, because none of these presumption are applicable, Claimant cannot establish the existence of pneumoconiosis pursuant to §718.202(a)(3).

Issue 2: Whether Mr. Jones' death arose from his coal mine employment.

Mr. Jones worked for over ten years in coal mine employment. Therefore, the presumption of causality provided for in 20 C.F.R. §§718.203(b); 718.302 is applicable. The record does not suggest any other employment that could be the cause of Mr. Jones' pneumoconiosis and Employer has not presented any evidence to rebut this presumption. Therefore, Claimant is entitled to a finding that if he had pneumoconiosis, that it arose out of coal mine employment. 20 C.F.R. §§718.203(b); 718.302.

Issue 3: Whether Mr. Jones' death was hastened by pneumoconiosis.

In addition to establishing the deceased miner had pneumoconiosis, in order to prevail on a claim for survivor's benefits under the Act, the claimant must also show that pneumoconiosis was a "substantially contributing cause or factor leading to the miner's

death." §718.205(a)(3). Death due to pneumoconiosis may be established under new §718.205(c) by one of the following criteria:

> Where competent medical evidence established that pneumoconiosis was the cause of the miner's death, or
>
> Where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or where the death was caused by complications of pneumoconiosis, or
>
> Where the presumption set forth in §718.304 is applicable.
>
> However, survivors are not eligible for benefits where the miner's death was caused by a traumatic injury or the principal cause of death was a medical condition not related to pneumoconiosis, unless the evidence established that pneumoconiosis was a substantially contributing cause of death.
>
> Pneumoconiosis is a "substantially contributing cause" of a miner's death if it hastens the miner's death.

§718.205(c)(1)-(5). Even prior to the introduction of the amended regulations, the "hastening death" standard was the rule in the Tenth Circuit. *Northern Coal Co.,* 100 F.3d at 874 (a survivor is entitled to benefits if pneumoconiosis hastened the miner's death "to any degree").

EIght physicians, Drs. Pearl, Randolph, Green, Perper, James, Naeye, Kleinerman, and Tuteur, rendered opinions regarding the cause of Mr. Jones' death.

Dr. Pearl signed Mr. Jones' death certificate and wrote that the immediate causes of his death were respiratory failure and idiopathic pulmonary fibrosis. (DX 5). Dr. Pearl's discharge report provided that his final diagnosis of Mr. Jones' illness was idiopathic pulmonary fibrosis and deep venous thrombus. Dr. Pearl's opinion, as reported in the death certificate and his discharge report, does not support a finding that pneumoconiosis contributed to Mr. Jones' death.

Dr. Randolph performed Mr. Jones' autopsy. His final diagnosis was that Mr. Jones has a clinical history of pulmonary fibrosis and increasing hypoxia with diffuse alveolar damage and pulmonary fibrosis. However, Dr. Randolph did not opine as to the cause of Mr. Jones' death. (DX 6). Accordingly, Dr. Randolph's autopsy report does not support a finding that pneumoconiosis contributed to Mr. Jones' death.

Dr. James opined that Mr. Jones' death was caused by respiratory failure secondary to his underlying lung and heart condition and that his respiratory failure was primarily caused by pneumoconiosis. (TX at 107). He also found that Mr. Jones had cor pulmonale and that the

cor pulmonale was a contributing factor to Mr. Jones' death. In his report, Dr. James also explained that it is not likely that the atypical pneumonia Mr. Jones had 1995 was a continuation of the pneumonia he had in 1982. In addition, Dr. James explained that there was a low probability that an acute infectious process was a causative factor in the development of his diffuse lung disease because there was a lack of a significant number of polymorphonuclear cells in most of the lung sections. (CX 6 at 2).

Dr. Green opined that Mr. Jones died "as a result of a progressive interstitial lung disease caused entirely by exposure to dust within the coal mines." (CX 1 at 5). Dr. Green based his opinion that Mr. Jones suffered from pneumoconiosis on several factors. First, Dr. Green found that previous pathologic reports had overlooked of misdiagnosed the presence of alveolar lipoproteinosis which he stated is a characteristic feature of the rapidly progressive form of silicosis. Silicosis is included in the Act's definition of pneumoconiosis. In addition, his examination of the lung tissue with a high powered microscope revealed needle-shaped particles which are characteristic of silica particles. Dr. Green clarified that although simple coal workers' pneumoconiosis usually does not progress without further exposure, this is not the case in miners who were exposed to high quantities of silica dust as Mr. Jones was. (CX 1 at 5). As Dr. Green explained:

> In summary, Mr. Clell Jones died as a result of a progressive interstitial lung disease caused entirely by exposure to dust within the coal mines. The high concentration of silicates and silica in this dust produced a less common, but well documented pattern of disease characterized by a predominantly interstitial pattern, together with alveolar proteinosis, indicative of a more acute and rapidly progressive response to silica dust. The pattern of fibrosis in the lungs did not in any way resemble that associated with UIP (or IPF).
>
> (CX 1 at 5).

Dr. Green rendered his opinion after reviewing Mr. Jones occupational and medical history, as well as his medical records and autopsy report. I find that his opinion is well-document and well-reasoned. Accordingly, I credit Dr. Green's opinion that Mr. Jones' death was hastened by pneumoconiosis.

Dr. Perper opined that Mr. Jones has an interstitial pulmonary fibrosis variant of coal workers' pneumoconiosis. He further concluded that Mr. Jones' death was caused by his diffuse pulmonary interstitial fibrosis of a rare variant of coal workers' pneumoconiosis which caused severe clinical impairment. (DX 7 at 8). He provided numerous studies in support of his medical diagnosis which I have previously credited. In addition, Dr. Perper rendered his opinion after reviewing Mr. Jones occupational and medical history, as well as his medical records and autopsy report. I find that his opinion is well-document and well-reasoned.

Accordingly, I credit Dr. Perper's opinion that Mr. Jones' death was hastened by pneumoconiosis.

Drs. Naeye, Kleinerman, and Tuteur all concluded that Mr. Jones' death was caused by severe interstitial fibrosis and not pneumoconiosis. In addition, all found that Mr. Jones' condition was not caused by his exposure to coal mine dust. Both Dr. Naeye and Dr. Kleinerman stated they found only minimal amounts of black pigment in Mr. Jones lungs. None of these doctors, however, used a high powered microscope to examine the lung tissue as Dr. Green did. Moreover, neither Dr. Naeye nor Dr. Kleinerman detected the needle-shaped particles which are characteristic of silica particles that Dr. Green found in Mr. Jones' lung tissue. I find that Drs. Green, Perper, and James' reports are more thorough than the reports of Drs. Naeye, Kleinerman, and Tuteur. Accordingly, I find that Claimant has met her burden of showing that pneumoconiosis hastened Mr. Jones' death in some way as required by 20 C.F.R. §718.205 and *Northern Coal Co.,* 100 F.3d at 874.

ORDER

IT IS HEREBY ORDERED THAT Employer shall:

(1) Pay Odessa Jones all benefits to which she is entitled to under the Act commencing as of the date of Mr. Jones' death.

(2) Pay Claimant's attorney, Jonathan Wilderman, Esquire, fees and expenses to be established in a supplemental decision and order.

A
PAUL H. TEITLER
Administrative Law Judge

Notice of Appeal Rights: Pursuant to 20 C.F.R. §725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board withing thirty (30) days from the date of this Decision by filing a Notice of Appeal with the Benefits Review Board, P.O. Box 37601, Washington, D.C. 20013-7601. A copy of this Notice of Appeal must also be served upon Donald S. Shire, Associate Solicitor for Black Lung Benefits, Francis Perkins Bldg., Room N-2117, 200 Constitution Avenue N.W., Washington, D.C. 20210.